

Valvular Heart Disease – High-Yield



1. Basic Concepts

Two major types of valve dysfunction

- **Stenosis:**
 - Valve **fails to open fully** → **obstructs forward flow.**
 - Usually due to primary cuspal abnormality (calcification, scarring).
- **Insufficiency / Regurgitation:**
 - Valve **fails to close properly** → **backflow.**
 - Caused by **intrinsic valve disease** (e.g., endocarditis) or **damage to supporting structures** (annulus, chordae, papillary muscles).

Valvular disease may affect one or multiple valves (mitral is most common).

2. Causes of Valvular Disease

A. Congenital

Bicuspid Aortic Valve

- Most common congenital valvular defect (**1–2% of live births**).
- Two cusps: **unequal size**, larger cusp has a **raphe** (incomplete separation).
- Function is normal in youth but prone to **early degenerative calcification** → **aortic stenosis**.

B. Acquired Valvular Disease

Main mechanisms:

1. **Degenerative changes**
 - Calcification (aortic cusps, mitral annulus).
 - ECM changes: ↑ proteoglycans, ↓ collagen & elastin (myxomatous degeneration).
 - Age-related due to mechanical stress (≈40 million beats/year).
2. **Rheumatic fever**
3. **Infective endocarditis**
4. **Other causes**

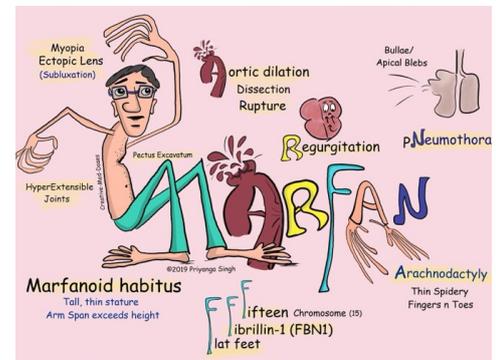
3. Important Acquired Lesions

1. Calcific Aortic Stenosis

- **Most common cause of aortic stenosis.**
- Parallel to **atherosclerosis** (similar risk factors).
- Often asymptomatic until severe.
- Calcifications restrict cusp opening → **LV hypertrophy, ischemia, → angina & CHF.**

2. Myxomatous Mitral Valve (Mitral Valve Prolapse)

- Non-inflammatory degenerative change of valve ECM.
- Linked to **type VI collagen defects**.
- Common in **Marfan syndrome**.
- Leaflets become **thick, floppy, enlarged with proteoglycan pooling**.



4. Infective Endocarditis (IE)

Definition

Microbial infection of valves/endocardium → vegetations of fibrin, platelets, and organisms with tissue destruction.

Common organisms

- **Streptococci**
- Staphylococci
- Fungi, rickettsiae, chlamydia

Common sites: **Mitral** & aortic valves; **tricuspid** in **IV drug abusers**.

Risk Factors

- **Preexisting heart disease**: RHD, HCM, VSD, bicuspid aortic valve
- **Degenerative valve disease**
- **Prosthetic valves, pacemakers**
- **IV drug abuse** (right-sided IE)

- **Pre existing structural heart disease**: rheumatic heart disease (RHD), hypertrophic cardiomyopathy (HCM), subaortic stenosis, ventricular aneurysm
- **Degenerative valve disease**: mitral valve prolapse, bicuspid aortic valve, calcific aortic valve disease
- **Cardiac interventions**: prosthetic valve, pacemaker, defibrillator, IVDA (right sided endocarditis), indwelling catheter
- **Congenital heart disease**: small ventricular septal defect, bicuspid aortic valve.

Pathogenesis

Two events required:

1. Endothelial injury → platelet/fibrin deposition
2. Bacteremia

Types

- **Acute IE:**
 - Highly virulent organisms, attack normal valves.
 - Severe, destructive, high mortality.
- **Subacute IE:**
 - Low-virulence organisms, affect abnormal valves.
 - Insidious onset, prolonged course; good prognosis with antibiotics.



Morphology

- Large friable vegetations on valves.
- Fresh lesions: **fibrin + platelets + neutrophils**.
- Chronic: organization, fibrosis, calcification.

Clinical Features

- Fever, rigors, night sweats
- New or changing murmur
- Embolic phenomena (brain → stroke; kidneys → hematuria; spleen → infarct)
- **Osler nodes, Roth spots**



Prognosis

- Fatal without treatment.
- Requires **long-term antibiotics (≥6 weeks)**.
- Take **3 blood cultures before starting antibiotics**.
- Valve replacement may be needed.

5. Libman–Sacks Endocarditis (SLE)

- **Sterile** vegetations in SLE.
- Due to **immune complex deposition** → **inflammation, fibrinoid necrosis** → **scarring**.
- Can appear anywhere on valve surface or endocardium.

Ultra-High-Yield Points

- **Bicuspid aortic valve** → early calcific aortic stenosis.
- **Calcific aortic stenosis** → LV hypertrophy → angina + CHF.
- **Myxomatous mitral valve** → Marfan; floppy leaflets.
- **IE requires: endothelial injury + bacteremia**.
- **Acute IE** = virulent organisms, normal valve.
- **Subacute IE** = low virulence, abnormal valve.
- **Osler nodes** = painful; **Roth spots** = retinal hemorrhage with pale center.
- **Take blood cultures BEFORE antibiotics**.



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"من كنوز الجنة"

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